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EDITORIAL

CAN THE DECLINE IN THE NUMBERS OF OUR HORSES BE ARRESTED?

As a passenger speeds along in an express train the countryside is laid before his gaze as are the changing colours in a kaleidoscope. There was a time when one of the prime attractions of this delight was centred in the horses which came successively into view. It was a veritable horse show in which all breeds were exhibited. There were horses in the fields, horses on the roads—they even invaded the railway, for they could be seen at work in the various sidings, or standing nonchalantly aside whilst the express sped by. There were horses of all types, sizes and breeds; good, bad and indifferent, and there were also the men in attendance to attract attention, for they all had something in common, the result of association or environment. They could be recognised at a glance as horsemen.

Times have changed and there is a sadness in the air, for on a recent journey of some eighty miles the number of horses which came within view could be numbered on one's fingers, and this sadness became a depression, for on a three-mile journey by road from the station not a single horse was seen. Later that evening a full realisation of catastrophy was revealed on reading a periodical, for it was written therein that, " Last year's returns indicated that there were less than 5,000 heavy horses under one year of age in the whole country."

What is the cause of this dreadful decline in the numbers of our horses? Is it the effect of mechanisation alone, or is it that we have failed our old friends? There is some precedent to support the contention that it is not all due to the simple inroad of the machine. When the railway network spread over the countryside about a hundred years ago horsemen were despondent, and there were those who said that the day of the horse was over. Indeed, this despondency was so great that when a branch line invaded his part of the

country, one relatively large owner stupidly disposed of his stock. But all these fears came to naught, for the railways stimulated a more voluminous and efficient transport system, and this, in turn, called for the use of more and more horses. At a later date the abolition of the tramway and omnibus horses gave rise to similar fears, yet the dire effects which were freely prophesied by some were not fulfilled if the prices fetched by the animals when they were put on the open market can be taken as a criterion of the demand.

There is no doubt that the recent diminution in the numbers of horses can be attributed in great measure to man's lack of interest. Gone are the days when the breaking and training of young horses was one of the joys and relaxations of the countryman, and when he who proved to be the most efficient in the art ranked supreme amongst his fellows. Such ambition and the spirit of emulation would appear to be satiated at the present time by the tone of a motor-cycle exhaust or a knowledge of the antics of the reigning cinema star.

The horses must not be allowed to go by this default. There is still a place for them on our farms and roads. Horses have been, and still are, a prime factor in our agricultural economy. Our breeds have achieved renown throughout the world, and justly so, for they are supreme in their respective spheres. We can do much to stimulate and revive the horse-breeding industry, and there is no need to bang the old drum of the comparative economies of horse and machine. Indeed, at the present time, with existing labour and wage problems, it is difficult to justify the use of horses on the grounds of economy alone. What is really needed is a revival of the inherent interest of the British people in the horse *per se*. Let us use all available means to stimulate the cult of the horse, and particularly apply our efforts to youth, for those who are of mature age need no such conversion. Young farmers' clubs, riding clubs, and the various breed societies are some of the agents through which we may carry our propaganda. If we can do this, and for the sake of our country we must do it, the present serious state of affairs may be arrested. If we stand mute then the decline will continue to an irretrievable end, an end which will bring dire consequences.

GENERAL ARTICLES

BRUCELLA ABORTUS INFECTION IN DAIRY CATTLE IN SINGAPORE

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A DAIRY farm under European management had been established on Singapore Island for many years and comprised approximately 200 acres of land with accommodation for nearly 500 imported exogenous dairy animals. The animals were imported from California and Australia, and they were mainly of Friesian breed. One of the official veterinary conditions of importation in the pre-war (1939) years was negative result to official double agglutination tests for *Brucella abortus*. This applied to animals imported from Australia and the United States.

No investigation had been carried out in Singapore before the war to establish the presence of brucellosis amongst indigenous animals as there appeared to be no indication that the disease existed.

The farm under discussion was conducted under strict rules of isolation and, as far as was possible, there was no contact with local animals. Animal feed used was imported from Australia and bulk feed comprised tropical fodder grasses grown on the farm (Guinea grass—*Panicum maximum*; and Napier grass—*Pennisetum purpureum*).

Despite these precautions the presence of brucellosis was suspected before the war, although this was never established by laboratory tests.

During the Japanese occupation, in 1942, the management of the farm was carried out by the Japanese on similar lines, although no records of management existed at the British reoccupation in 1945.

In 1945, only a handful of animals remained, as the Japanese had destroyed nearly the whole herd prior to the British reoccupation. These remaining animals were kept for manure production for fodder grass rehabilitation and appeared to be in good condition. No tests for brucellosis were possible at that time.

In late 1946, the owners of the farm decided to restock with Fresians imported from Australia, and, as a safety measure, all the remaining pre-war animals were destroyed before new animals arrived. Importations continued up till April, 1949, in batches from Australia. All animals were negative to official Australian double agglutination tests, as required by the existing Singapore Veterinary regulations (see appendix). These regulations did not cover New Zealand, as up till then no importation from that country had occurred.

Prior to April, 1949, a few animals had aborted, and this was not considered of particular importance in view of the long sea voyage to Singapore, and agglutination tests carried out after a month of these animals failed to detect any positive reactors. No herd tests were carried out up till 1949.

In April, 1949, a batch of 38 in-calf heifers arrived from New Zealand and, as the official Veterinary legislation did not cover importations from New Zealand, the animals were landed, and after a period joined the main herd. Twenty-six of these animals had not been subjected to double agglutination tests, but had received S.19 vaccine at varying ages.

This fact was brought to our notice after a delay of a few weeks, and some concern was felt as there appeared to be no evidence that inoculation with S.19 vaccine also prevented the animals from carrying the virulent organisms and causing disease among susceptible animals. The fact that the animals had received S.19 vaccine presupposed that they had come from an area or farms in which contagious abortion existed.

The owners were advised to subject the whole herd to a series of agglutination tests, and this was carried out (see Tables I-V). At the same time a number of indigenous Zebu cattle from other small licensed Indian dairies were tested similarly. Twenty-one animals belonging to a Government cattle farm in Johore, approximately 100 miles away, were also tested, as a few abortions had been recorded there (see Tables VI and VII).

Agglutination Tests

TABLE I

September, 1949.

The antigen used in this test was supplied by the Animal Research Station, Wallaceville, New Zealand, as "*Brucella abortus* antigen (Concentrate)," sent here by air mail in refrigeration.

	No. of animals	%
No agglutination at 1/12.5 or higher	60	24.1
Agglutination at 1/12.5	90	36.1
Agglutination up to and including 1/25	47	18.9
Agglutination up to and including 1/62.5 ...	12	4.8
Agglutination up to and including 1/125 ...	6	2.4
Agglutination up to and including 1/250 ...	34	13.7
	249	100.0

The high percentage, nearly 40 per cent, of suspicious (positive agglutination up to 1/25) and definite reactors (titres greater than 1/25) gave rise to great concern, and further tests were carried out to confirm the above results. Steps were also taken to isolate the organism from any abortion that occurred.

The antigen used in subsequent tests was supplied by the Ministry of Agriculture and Fisheries Veterinary Laboratory, New Haw, Weybridge, U.K., as "Standard *abortus* antigen (Concentrate)."

TABLE II

November-December, 1949

	Immunised.	Not immunised.	Number of animals.	% of 339.
No agglutination at 1/10 or higher ...	6	115	121	35·7
Agglutination at 1/10	5	87	92	27.1
Agglutination up to and including 1/20 ...	5	48	53	15.6
Agglutination up to and including 1/40 ...	7	21	28	8.2
Agglutination up to and including 1/80 ...	3	17	20	5.8
Agglutination up to and including 1/160 ...	0	25	25	7.6
	26	313	339	100.0

TABLE III

January-February, 1950

	Immunised.	Not immunised.	Number of animals.	% of 345.
No agglutination at 1/10 or higher ...	7	150	157	45·5
Agglutination at 1/10	9	63	72	20.8
Agglutination up to and including 1/20 ...	2	42	44	12.7
Agglutination up to and including 1/40 ...	7	20	27	7.8
Agglutination up to and including 1/80 ...	0	11	11	3.2
Agglutination up to and including 1/160 ...	0	34	34	10.0
	25	320	345	100.0

The Ministry of Agriculture and Fisheries regard those animals having a titre of 1/40 or greater as having "failed the test for non-agglutinins." Titres of 1/20 are regarded as indefinite or suspicious, while titres of 1/10 or below would "pass the test for non-agglutinins."

Thus we have Table IV:—

TABLE IV

	September, 1949. %	*November*, 1949. %	*January*, 1950. %
Non-reactors ...	60.2	62.8	76.3
Suspicious	18.9	15.6	12.7
Definite reactors ...	20.9	21.6	20.0
Number of animals	249.0	339.0	345.0

These figures are not strictly comparable as the herd population had changed between tests. Some animals had been slaughtered, some animals were not included in the tests for various reasons, and new animals had been added to the stock. Nevertheless, while the overall percentage of definite reactors remained constant at about 20 per cent, there was a shift in the number of

those classified as suspicious towards negative, probably due to new non-reactors being added to the herd.

By far the most interesting result is obtained by comparing the titres of animals tested twice. In the following table all the animals had been imported under " the double agglutination test " (see Appendix).

TABLE V

Results of Two Tests

Number of animals: 293, all previously certified agglutinin-free

Results of tests in Nov.-Dec., 1949	Results of tests in Jan.-Feb., 1950							Totals Nov.-Dec., 1949	%
		0	1/10	1/20	1/40	1/80	1/160		
0	...	84	14	3	4	0	0	105	36.1
1/10	...	37	29	11	2	0	1	80	27.3
1/20	...	11	16	14	2	1	3	47	16.0
1/40	...	2	1	7	10	0	2	22	7.5
1/80	...	0	0	0	1	7	6	14	4.7
1/160	...	0	0	0	0	3	22	25	8.5
Totals Jan.-Feb., 1950	...	134	60	35	19	11	34	293	
%	...	45.7	20.5	12.0	6.5	3.7	11.6		100.0

The results indicated a high proportion of infection in the herd, but there was no clinical evidence to support this conclusion. The actual incidence of abortions throughout the period of investigation was not higher than could be expected on previous experience and certainly did not approach that of established contagious abortion.

Six of the results in Table V were of particular interest, viz., the titres which jumped from 1/10-1/40 to over 1/160. One of the cows which showed these reactions later aborted and from the fœtus *Brucella abortus* was isolated (*vide infra*).

TABLE VI

Results of Tests on Indigenous Dairy Cattle

	No. of animals	%
No agglutination at titre of 1/10 and above ...	26	81.3
Agglutination at 1/10	4	12.5
Agglutination up to and including 1/20	1	3.1
Agglutination up to and including 1/40	1	3.1
Agglutination over 1/40	0	0
	32	100.0

TABLE VII

RESULTS OF TESTS ON JOHORE CATTLE—SUSPECTED TO BE INFECTED

	No. of animals	%
No agglutination at titre of 1/10 and above ...	14	66.7
Agglutination at 1/10	2	9.5
Agglutination up to and including 1/20	0	0
Agglutination up to and including 1/40	1	4.8
Agglutination up to and including 1/80	2	9.5
Agglutination up to and including 1/160 ...	2	9.5
	21	100.0

Table VI showed that infection, as far as agglutination tests could detect, was not evident in the indigenous dairy farms.

Table VII, on the other hand, indicated that suspicions of infection in certain Johore cattle were well founded, and *Brucella abortus* was later isolated from one of these cases (*vide infra*).

Isolation of Brucella abortus

The medium used was liver infusion agar with gentian violet 1/250,000. For sub-culture of suspicious colonies, liver infusion agar without gentian violet was used. Incubation was at 37° C. with 10 per cent CO_2 and in atmospheric oxygen.

The alimentary tract was removed entire from the fœtus and sent to the laboratory with spleen and liver and heart. In the laboratory, after usual precautions cultures were taken from stomach contents, spleen and liver. Guinea-pigs were inoculated subcutaneously and intra-peritoneally with stomach contents and emulsion of spleen. Direct smears of stomach contents and spleen and liver were also examined.

Brucella abortus was isolated from two cases.

The first case was from an aborted fœtus of seven months' gestation from the Singapore farm. Due to the prompt action of the dairy farm management the organs, etc., were received the same day and were in satisfactory condition. An organism resembling morphologically *Brucella abortus* was seen in small number in direct smears of stomach contents and spleen. The organism was isolated in culture from stomach contents and spleen. Guinea-pigs inoculated with stomach contents and emulsion of spleen both showed, after six weeks, typical pin-point necrotic lesions in liver with ischæmic enlargement of lymph nodes. The organism was recovered in culture from the spleen of the guinea-pig. It corresponded morphologically, culturally and biologically to *Brucella abortus*.

Subsequent to this case from Singapore, *Brucella abortus* was isolated from materials sent by the Johore Veterinary Department. The fœtal stomach and vaginal swabs from a case of unexplained abortion in a buffalo were received. Cultures, smears and inoculation of guinea-pig from fœtal material were negative, but *Brucella abortus* was isolated from the vaginal swab.

Comment

1. The attempt to prevent by legislation the introduction of brucellosis from abroad into the colony has failed for reasons beyond official control.

2. The source of infection remains a matter of conjecture, as the agglutination picture prior to April, 1949, was not available apart from the initial entry of animals under double agglutination test. There is a possibility that infection may have fulminated at the premises throughout the Japanese occupation, particularly as heavy dressing with manure was a necessary adjunct in obtaining the required heavy fodder grass yields for so many cattle. The possibility of spread of infection from local cattle is unlikely in view of the isolation of the property and there being no sign of infection among the small numbers tested (32 head). It is also possible that the infection had been imported more recently. Animals which had been found agglutinin-free could have been infected between the time of testing and arrival in Singapore, though this should amount to unwarranted carelessness on the part of the shippers. The non-reactor which excreted virulent *Brucella abortus* has also been reported.[1] Infection from the immunised animals imported from New Zealand is the remaining possibility.

3. The relatively high percentage of positive reactors and the small number of animals that have aborted in the initial stages of introduction of the disease into the herd may be followed by a higher abortion rate in the immediate future. Sir Weldon Dalrymple-Champneys has stated[2]: "On the first introduction of the disease into a herd, abortions usually occur only at long intervals and the infection is seldom detected for a considerable period." The detection of infection in our case has enabled epidemiological precautions to be undertaken. At the time of writing there is still no clinical evidence of an outbreak.

4. The commitments of the company are such that should the abortion rate rise, the difficulties of maintaining milk supplies and the expense of stock replacement from Australia are of primary consideration. They are unwilling to carry out the orthodox method of elimination of reactors and the building up of an "abortus-free" herd, particularly in view of the difficulty of carrying out an isolation policy on such a restricted property. The use of S.19 has now been adopted to the whole herd at least as a temporary expedient of insurance against economic loss.

Summary

1. The results of agglutination tests on a herd of imported dairy cattle indicated *Brucella abortus* infection.

2. Except for very occasional abortions there was no clinical evidence of the disease.

3. Infection at the farm was established by the isolation of the organism.

Acknowledgments

We wish to thank Mr. W. Corrigall, M.R.C.V.S., for his valuable help and co-operation, and Mr. C. C. Renwick, M.R.C.V.S., the State Veterinary Officer, Johore, for access to clinical materials and keen interest.

APPENDIX

"Its [the animal's] blood shall be submitted to two such [agglutination] tests at an interval of not less than one month between each test with negative results in both cases and provided that the second and last test shall be carried out not more than one month prior to export."—Singapore Veterinary Quarantine Rules, 1937.

REFERENCES

(1) Topley and Wilson: "Principles of Bacteriology and Immunity," Third Edition, 1946, p. 1715.
(2) Sir Weldon Dalrymple-Champneys: Milroy Lectures, 1950, *Lancet*, 1950, I, p. 429.

ELECTROPLECTIC ANÆSTHESIA IN ANIMALS AND MAN

By E. O. LONGLEY, B.Sc., M.R.C.V.S.

(Concluded)

In consideration now of the electrical requirements, the variables involved in the problem as constituted are the e.m.f. and regulation of the source of power, these deciding the p.d. across the subject according to the latter's (indeterminate and changing) resistance, which in turn decides the flow of current; the electric wave-form and its frequency; the site of application (and size) of the electrodes; the manner in which the current is passed (pattern of increment and stability); the duration of application; the species and size of animal; and idiosyncratic differences between individuals. In the circumstances it is perhaps hardly surprising that such little knowledge as exists is somewhat tenuous and indifferently established, or if the existing procedures are rather hit-or-miss. While a certain amount of information may be gleaned from medical studies and practice of the method in man, these are data derived in a very different venue and context from the veterinary, and it may be said that really there is no representative body of comparative knowledge anywhere in the matter.

A first necessity is obviously simplification of the problem by the elimination of as many of these many variables as possible, and for practical reasons that have been indicated, sine-wave current from the mains at 50 cycles may be taken as a first standard of reference and a first constant. But the priority requirement, more important than any, is unquestionably the provision of constant-current conditions. Reference has been made in a separate related paper (Longley, 1950a) to the difference between constant-voltage and constant-current conditions and the physiological desirability of the latter. It cannot be overemphasised that voltages are contextually only of indirect interest, and, indeed, in themselves convey very little. In fact, with good regulation at the source a voltmeter across the subject can give much the same reading when the animal is

receiving its full intended dose as when there may be open-circuit and no current whatever is passing, and for all values of current between. Voltage data can, accordingly, be grossly misleading, yet they are the most universally adopted standard of contexual reference, as witness the vast majority of the literature here cited. It requires emphasis and re-emphasis *that it is current that is the ultimate determinant of events, and ammeters that provide the only data really informative.*

These considerations define the fallacy of the customary constan-voltage operating conditions, as with volts from the mains stepped down to a more or less constant 70 volts. For this is to render the " inconsequential " factor voltage stable, while the supremely consequential factor *current* is made an unpredictable variable governed by the fortuitous circumstances of the individual case. This is the very reverse of the true and obvious requirement. It is therefore only the more surprising that both in the ordinary routine of electroplexy in man and in the abattoir, constant-voltage conditions should prevail, with all their attendant obscurantism and disadvantage. In contrast, with constant-current condition (the provision of which need present no special technical difficulty), the widest variations in the resistance of the skin, etc., of the subject make negligable difference to the current passing. Constant-current conditions must, accordingly, be recognised as the first pre-requisite of the case, viewed from whatever angle. This is, in effect, to substitute current as a single predeterminable constant variable at will, for at least six of the complexly interdependent variables listed.

A further variable was earlier dealt with by the statement that the current should traverse the cerebrum. Two further identities, size and idiosyncrasy, may for the practical purpose in view be unified. Because for reliable anæsthesia, interest is necessarily and narrowly focused *on the requirements of the electrically resistant subject.* If these are not met, whatever may be the proceduce must be anæsthetically inefficacious in a proportion of animals; while if they are met, the needs of the rest are *ipso facto* provided. The essential requirement for sound practice is manifestly less a knowledge of the *low* threshold for convulsions at various weights than of the *high* threshold and the requirement of the idiosyncratically resistant subjects of the various species and weights. And it emerges that whereas in the abattoir the adopted policy has been to employ current at the nearest possible approach to the lowest effective level, the prescription for reliable anæsthesia is the very reverse—the use of current at whatever higher level (in consideration also of the time of application) may be necessary for universal efficacy. It is also the very unsatisfactory position that, despite the use of electroplexy on animals totalling many millions, the requirements in question are unknown.

Summarising, the legitimate practical requirements are seen to be:—

(1) The provision of constant-current conditions.

(2) Knowledge of the strength-duration requirements of the electrically resistant of each species, considered in relation to size.

(3) The provision of apt methods of applying the electrodes, appropriate of each species.

(4) The provision of such modifications of technique, in whatever direction, as may be necessary for the elimination of all significant risk of secondary effects, with particular attention to the *glissando* technique.

This last refinement is procurable either manually, as in human electronarcotic practice, or better by automatic means, either mechanical (*cf.* the automatic telephone dial), or electrical, as employed in the present studies.

A final consideration requiring mention is the availability of electric power, particularly in veterinary field work, and where there may be no electric mains. A solution to this is immediately offered by the power available in the ordinary car battery, which is ample for the purpose. The basic requirement for constant-voltage conditions is not more than the conversion of 6 or 12 volt d.c. into something probably not exceeding 100 volts a.c., with a sufficient wattage. This requires only a rotary-convertor or motor-generator to be plugged in on the car, and it is a requirement which has in fact already been met for medical purposes, by the manufacture of E.C.T. apparatus to run from car batteries for use by the doctor in country districts. With a sufficient length of cable the method would thus become available in every ordinary circumstance of veterinary practice. It may be added that in consideration of portable apparatus, the high potency and consequent low current demands of square-waves, discussed earlier, could additionally be exploited to advantage.

It should be clearly represented that these concepts do not threaten complicated apparatus and difficult, critical operation. On the contrary, the objective specifically in view is the elimination to the maximum of the human element with is capacity for error, and the provision of self-operating, simple equipment at low cost, adjustable to the requirements of each case in terms of reliable data, and generally operable as simply as a radio set may be tuned in, and as is certainly possible by reason of the fundamental simplicity and wide margin of safety of the method.

General Discussion

It is difficult to understand why the obvious advantages of E.P.A. in veterinary clinical work have remained so long in the limbo of such neglect. Possibly the widespread use of the method for slaughter, and the general adoption of the term "electrolethaler," with its entirely misleading implication that the electricity is the lethal agent, has militated against an understanding of the realities. Hobday, himself (1935), writing as an advocate of the clinical adoption of the method, is responsible for the very paradoxical remark: "There were no after-consequences and the pigs took *the lethal dose** without the slightest trouble." As it is hoped has been amply demonstrated, the lethal agent is the slaughterman's knife, and the proper designation of the instrument is the "electro-anæsthetiser."

* My italics.—E.O.L.

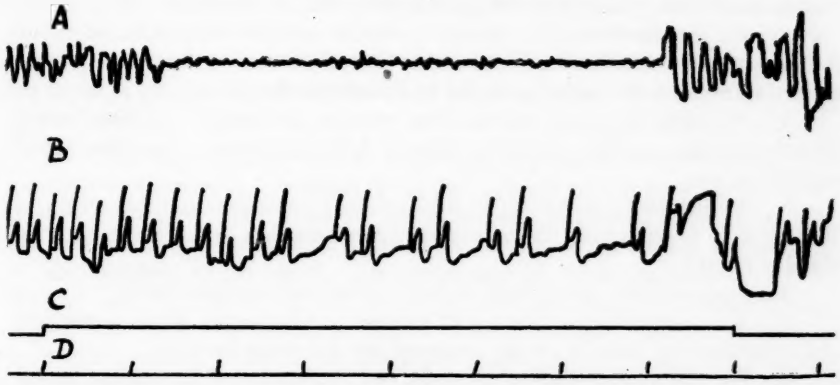
Whether the feature of the convulsion may ever have been considered a disqualification of the method for clinical use, on æsthetic grounds, can hardly be known, but if so it is an attitude which certainly cannot be approved. Struggling, particularly when violent, is more objectionable æsthetically in the conscious animal under forcible restraint, as in the cat undergoing castration without anæsthesia. It is, moreover, constantly featured during the induction of ordinary inhalation anæsthesia, and then for considerably longer periods than with E.P.A. As the signal of unconsciousness, the convulsion of E.P.A. joins the company of the corneal reflex and the other indices of unconsciousness, and is to be regarded in this light. It may be said, however, that with an appropriate technique it should be possible to control the convulsion electrically, either partly or completely, at will. This statement is based on the American routine for the so-called electro-narcosis in experimental dogs and in man, and observations in the present studies on the rabbit. Such a technique would, however, be contingent on a sufficiently complete knowledge of the requirements, comparative and otherwise.

The clinical possibilities have been detracted from also in the topical literature, as where Clark and Tweed (1932) observed: "A suggestion has been made that the apparatus [the 'electro-lethaler'] may be used to produce unconsciousness in animals for minor operations, but in view of the enormous strain on the circulatory system, the wisdom of this suggestion is doubtful." Whether this understandable apprehension was in relation to cardiac or vascular consequences, the experimental data here adduced, no less than the liberal use of the method on senile and necessarily arterio-sclerotic human subjects, and on those with even serious cardiac insufficiency, must now allay such misgivings.

As has already been mentioned, doubts have been expressed in the literature as to the fundamental validity of E.P.A. (Hertz, 1933; Roos and Koopmans, 1934, 1936). Indeed these authorities go so far as to state categorically that the anæsthesia is spurious, being a condition of motor-paralysis simulating anæsthesia ("En réalité . . . c'est un cochon stupéfié, mais ce n'est pas un cochon anésthésié. Le bénéfice est certain pour les spéctateurs émotifs, il est nul pour le cochon" (Hertz, *op. cit.*). Hertz's views are formed by somewhat false analogy with certain experimental observations of his own on human patients in a different but related context, which are themselves not above a certain suspicion, as is discussed elsewhere (Longley, 1950b). The views of all three observers are formulations anteceding the large-scale use of electroplexy in psychiatry, and instance almost classically the desirability of caution in affirming negatives in such fields as this. The evidence since accumulated from human subjective experience completely vitiates their contention, at the same time as it indicates that the anæsthesia *can* be fictitious, in the terms already discussed. But that underdosage can determine imperfect anæsthetization is neither surprising nor remarkable.

Before leaving this aspect, reference should be made to the question, raised by these several critics, of the validity of tests for unconsciousness in the circumstances, a matter which has received previous brief consideration elsewhere

(Longley, 1949). The writer's previous observations may now be amplified to the extent that it is necessary to distinguish sharply between (a) those circumstances where the current is still in transit, and those where it has ceased to pass; and (b) in the latter respect, between the situation before the onset of the convulsion and that after the convulsion. While current is passing, and thereafter until the onset of the convulsion, the state of consciousness may not be reliably ascertained by any known objective means, owing respectively to the indeterminate central and musculo-tetanic effects of the current, and the possibility of frank motor-paralysis. Tests for consciousness are essentially dependent on integrity of the effector side of the reflex arcs and on a constant correlation between the deterioration of the reflexes and consciousness, and as neither is reliably featured contextually, the case differs essentially from that with chemical anæsthesia. This is an issue which has been quite inadequately acknowledged and investigated with the use of electricity. The principle question remaining at issue with E.P.A. however is that of the duration of the anæsthesia and whether there is any possibility that the subject may, *post-convulsively,* be conscious and receptive of sensory stimuli but



Record illustrating apnœa and bradycardia consequent on the exhibition of amyl nitrite vapour to the rabbit. *A,* electropneumogram; *B,* electrocardiogram (thoracic leads); *C,* signal; *D,* time in seconds. The irregularities in the E.C.G. result from movement by the animal. Even considerably greater slowing of the heart may be produced by this means without apparent central effects (see text).

in a condition of retained motor-paralysis as is possible *pre*-convulsvely, as has been mooted. It may be said at once that with electroplexy there is no reason to suppose that this can occur. The reverse is rather to be expected, in accordance with the greater complexity of the machinery of connation, and the dazed and confusional states which prevail some time *after* the recovery of motor-power, and which are to be observed also in animals. And satisfactory evidence in context was afforded to the writer by personal observations at the leucotomy operation in man,** where with the operation still in progress subdued defensive movements may be seen, with lightening of the anæsthesia, considerably before there is any question of recovered consciousness and awareness. Thus it may be

**By kind invitation of Professor Golla.

confidently inferred that in animals also motor indications of recovery, well in advance of the recovery of consciousness, are available and manifested as indices of the duration of anæsthesia (as with lightening of ordinary chemical anæsthesia).

Considering E.P.A. in its veterinary clinical applications, it is abundantly evident that the method is 100 per cent safe anæsthetically, and could be used on dogs and cats without any apprehension of primary or secondary accident; that in the larger animals, particularly the pig, while basically as safe, there is room for improved techniques, when it should become equally safe in secondary ways. Whether it is also applicable in the adult ox and the horse remains an open question.

The special purposes for which it might serve are:

1. As the sole anæsthetic for essentially brief procedures of all kinds.
2. For the induction of general anæsthesia, as the preliminary to maintenance anæsthesia by ordinary chemical means.
3. For immobilisation in radiography, instead of the customary intravenous anæsthesia (fractures unfortunately excluded, in general).
4. For ophthalmoscopy, where it would combine the advantages of midriasis with immobility, and at the same time eliminate the delay and persistence of effect with the use of atropine or homatropine.
5. For other physical examinations, notably abdominal palpation where there is excessive guarding (again in place of I/V anæsthesia); deep pharyngeal conditions; etc.
6. Sometimes for handling, for the more easy application of other measures of control, in exceptionally difficult or dangerous subjects (a use emphasised by Hobday (1935)).
7. Exceptionally, as a simple sedative, administered repetitively.
8. For anæsthesia in captive wild animals in menageries, which possibility, also recognised by Hobday, is not proposed for discussion here.
9. For euthanasia, whether by prolonged application of the current, or for anæsthesia to be followed by intrathecal (intramedullary) injection, by which latter means it would be possible to destroy any animal absolutely painlessly, with absolute certainty, at no cost, in appreciably less than a minute.

The induction of E.P.A. is seen to require at the most 45 seconds over all, and nobody could maintain that this is an exorbitant time. The duration of full anæsthesia in the domestic animals is on the available evidence probably one to two minutes. On certain of the evidence it may be that longer periods are possible, but even one or two minutes is time enough and to spare for any number of the requirements in clinical work. And where more time is demanded there are no evident prohibitions to repetition of the procedure. In circumstances where large numbers of animals are involved even this expedient method may be adjudged time-consuming (and unnecessary) by some, though what is expeditious enough for the high-pressure work at the abattoir should suffice for anything. In fact, however, the method properly developed technically would be automatic and fool-proof, operable by any intelligent lay assistant

under supervision after 15 seconds' instruction, so that no time need be lost except for the 45 seconds for the first of the series. And the writer would like in any case to register this mild protest that the humane standard in veterinary clinical practice in certain contexts, should lag by two decades on the ethical standards of the slaughterhouse. After all, the bleeding-out process is painless, and the sticking process itself certainly less painful than many clinical procedures traditionally accomplished in veterinary work without any pretention to anæsthesia.

On the other hand, notably in large pigs, boars and nursing sows; no doubt potentially in the bull, steer, heifer, cow, and possibly also in horses; and in certain contingencies of canine and feline management, the adoption of this measure would save time over and again by eliminating the problem of control, which is so often the only difficulty in a task of seconds. There can be no need to enumerate its many applications, but obvious appropriate contingencies are castrations, spaying (especially gilts), ringing the nose, dental work, the incision of abscesses, tattooing and branding, debudding, dehorning if the method is serviceable in adult cattle, etc., etc. Or generalising, in the majority of those circumstances where forcible control, with or without local anæsthesia, is the present custom; and at other times in place of unnecessarily prolonged and more dangerous forms of general anæsthesia, the use of which is legitimately avoided as much as possible.

Nor do these occasion exclude certain painful procedures and routine requirements or research, as opposed to clinical work. Every research worker of experience will agree that the animal which, through repeated painful treatments has become conditioned to resist all handling, is as much a burden and liability to the worker as to itself. The existence of such animals is a reflection and indictment of our incompetence, and of the inadequacy and impropriety (with alternatives available) of anachronistic practices sanctioned by a bad tradition. Humane considerations quite apart, it is objected that the creation of such situations is properly and simply definable as grossly inefficient.

Whether E.P.A. is in fact practicable in adult cattle and the horse is hardly likely to be known unless it is essayed. There do not appear to be any very convincing reasons why it should not be. Crush-fracture is the obvious risk, more particularly in equines, but sufficient has been said on the implications of this and its possibilities of technical evasion. It may be remarked that crush-fracture is commonly associated in ordinary casting procedure with an *emprosthotonic* posture, and that this is the reverse of that characteristic of electroplexy. From the musculo-skeletal aspect a legitimate analogy can be drawn with the situation in tetanus, and there does not seem to the writer to be any significantly greater probability of the complication in question with E.P.A., than in tetanus or indeed than with ordinary casting in courageous animals. It is of course in the large animals that the initial flexion of the hind legs would be valuable, to bring them into recumbency. And also that the designated electrical control of the convulsion would have special recommendations. But conjecture is idle at

all such points, and practical trial is as ever the only means to knowledge in the matter.

Fifteen years ago the late Sir Frederic Hobday, writing of clinical electroplectic anæsthesia as a veterinary surgeon uniquely experienced and eminently practical, declared: "In my opinion this method has a very valuable practical future." No doubt Professor Hobday did not anticipate that his foresight would first be represented in the human operating theatre. His visionary concept the present writer cannot sufficiently endorse, and it is submitted that the veterinary profession should renounce its past policy of mistrust and neglect of these rich possibilities, and follow the lead at last offered it in a field of human surgery.

Summary

1. Electroplectic anæsthesia is defined and its features described and discussed. The importance of the convulsion as an index of satisfactory procedure is emphasised.

2. The existing practices are outlined and criticised, and the requirements designated.

3. The recent use of the method for surgery in man is noticed. With correct techniques based on knowledge of the comparative requirements, it could be made available for veterinary clinical purposes as a means of instantly induced anæsthesia, automatically operated, completely reliable, and probably 100 per cent safe.

4. The veterinary applications of such a method are nominated, together with the evident contra-indications. Its special promise of convenience for short surgical measures of all kinds; for the induction of anæsthesia prior to maintenance by ordinary chemical means; and for handling troublesome subjects, is emphasised.

Acknowledgments

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EXPERIMENTS WITH DRIED BRACKEN (PTERIS AQUILINA)

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BRACKEN POISONING is a serious hazard in cattle husbandry on hills where bracken grows in abundance. In some parts of the country it also occurs in horses. The first description of bracken poisoning is that of Storrar (1893), and since that time many clinical and experimental observations on both cattle (Stockman, 1917) and horses have been recorded. Reviews of previous work are given by Shearer (1945), Boddie (1947), Stamp (1947) and Evans & Evans (1949).

Recently, attention has been focused on the anti-thiamine factor of bracken. This was first demonstrated by Weswig, Freed & Haag (1946), who produced thiamine deficiency in rats by feeding a diet containing 40 per cent dried bracken; the anti-thiamine activity was not decreased by air drying at 105° C. for 18 hours and the factor was slightly soluble in 92 per cent ethyl alcohol but insoluble in ethyl ether and acetone. Confirmation of the presence of an anti-thiamine factor was obtained by Evans & Evans (1949) and Thomas & Walker (1949). The former noted that rats fed dried bracken had a low vitamin B_1 and a high pyruvic acid content in the blood and that aqueous extracts of bracken fronds and of the rhizomes inactivated vitamin B_1 *in vitro*. Thomas & Walker (1949) in addition found that the fæces of rats fed bracken still contained the anti-thiamine factor, and suggest that it is not absorbed but that it acts on the vitamin B_1 of the food while it is in the alimentary tract. Since then a preliminary report of bracken poisoning in a horse which responded to B_1 therapy has appeared (Roberts, Evans & Evans, 1949), and two unsuccessful attempts to treat bracken poisoning in cattle with vitamin B_1 have been reported (Vaughan & Roberts, 1949; O'Moore, 1949).

A preliminary report of biochemical studies on the anti-thiamine factor has been made and it appears that this factor splits the thiamine molecule into its two components, pyrimidine and thiazole (Evans, Jones & Evans, 1950). It is probable that the presence of this highly active material in bracken and the onset of bracken poisoning in cattle are related, but the nature of the cattle syndrome and the possible complications presented by the rumen do not lend themselves to the conception that bracken poisoning is a straightforward vitamin B_1 deficiency and more work is necessary in order to clarify the position.

The experiments recorded here constitute a preliminary investigation into the problem and form the basis for a larger experiment in which adequate numbers of animals are to be used.

Method

The main object of these experiments was to find a way of gathering and feeding bracken to large animals which could be relied upon to produce " active "

bracken in the sense that it would produce bracken poisoning. Bracken poisoning has not been reported in the North of Scotland and for this reason it seemed unwise to rely entirely on local bracken, but the cost of harvesting large quantities of bracken from a known " bracken poisoning area " would have been considerable. After consideration of the information available, it was decided to gather a large harvest of bracken locally and a small quantity of bracken from a known bracken poisoning area, so that a comparison of the two samples could be made on rats while local bracken was fed to large animals. Tests in a hot-air tunnel showed that whole bracken fronds needed 2-3 days to dry, and as the amount that could be placed in the tunnel was enough to feed one steer for one day this method was abandoned in favour of a grass drier. A trial with a grass-drying plant showed that if bracken was chopped in a silage cutter it would dry satisfactorily. The following procedure was therefore adopted :—

Bracken was cut during the early stages of growth when the top few fronds had opened. It was transported to the silage cutter and chopped. It was then taken to the grass drier and dried with a minimum of delay. The dried material was ground to a powder and stored in paper sacks. The time-table of these operations was as follows :—

June 16 and 17.—Bracken cut by scythe and stacked in heaps. Carted to the farm on the afternoon of the 17th.

June 18.—All bracken chopped in a silage cutter and taken to the grass-drying plant. 9 p.m., drying started and continued until 10.30 a.m., June 19, with a few hours' break in the early morning.

Finally the material was ground and stored in paper sacks.

Weight of bracken cut	...	...	120 cwt.
Weight of dried bracken	...	...	30 cwt.

A small sample of bracken was picked by hand from 9 a.m. to 12 noon on 24/6/49 at Buckholm, Galashiels, an area where bracken poisoning has occurred. Only the top leaves and the tips of the stems were taken. The bracken was dried in a Templewood drier between 1 and 2.30 p.m. the same day. About 14 lb. of dried bracken was ground in a hammer mill. The remainder was not dry enough to powder, and was bagged as it was.

The material was taken to the Institute by rail and arrived at 10 p.m., June 24. The semi-dried material was later dried in a building by turning daily and was bagged on 28/6/49.

Experimental Results

The dried bracken so obtained was fed to rats, ponies and cattle, and the results are presented in that order.

1. Experiments with Rats

Experiment 1.—Six litters of weanling rats from the Rowett Research Institute colony were used, 3 males and 3 females being taken from each litter except one which contained only 1 male. The rats were caged individually on raised wire floors and given no bedding. They were given food and water *ad lib.*

Three diets were used which had the following composition:—

			Diet A	Diet B	Diet C
Casein (Glaxo C.)	12	Basal diets			
Lard	8				
Dried brewers' yeast (Pharmaco-Chem. Co.)	6		60	60	60
McCollum's salt mixture	5	60 parts			
Cod-liver oil	1				
Sucrose	28				
Dried bracken (local)			40	—	—
Dried lucerne meal			—	40	—
Casein (Glaxo C.)			—	—	5
Glucose			—	—	35
			100	100	100

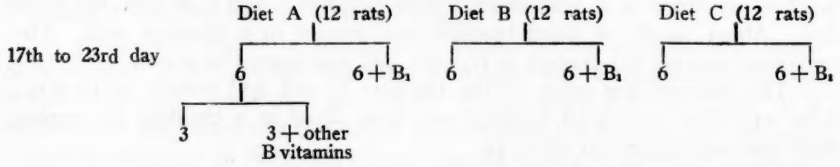
It was necessary to moisten diets A and B as when dry the rats scraped it out of their pots.

From the 17th to the 23rd day of the experiment half the rats in each group received daily intramuscular injections of 20 μg. of vitamin B_1 hydrochloride on six occasions.

Later, 3 rats receiving diet A were given intramuscular injections of 0.5 ml. of an aqueous solution of B vitamins other than B_1. Each dose consisted of:—

100 μg. Ca pantothenate
15 μg. Pyridoxine
15 μg. Riboflavine
0.5 μg. Biotin
15 μg. Pteroylglutamic acid

The plan of the experiment thus is as follows:—



All rats survived the experimental period of 5 weeks, but growth on diet A (bracken) was less than on the other two diets. The injection of vitamin B_1 had a significant growth-promoting effect on the rats fed on diet A, but no effect on rats eating diets B and C. This is illustrated by Fig. 1. Growth curves of males and females were not significantly different. No signs of vitamin B_1 deficiency other than a depression of growth rate were seen in rats receiving diet A and the heart rate of these animals as measured on a Cambridge Instrument Co. electrocardiograph showed no difference between the groups.

The administration of B vitamins other than B_1 had no growth-promoting influence.

Table I shows that as far as can be judged by the analysis of the lucerne meal and local dried bracken there is little to choose between them as regards food value.

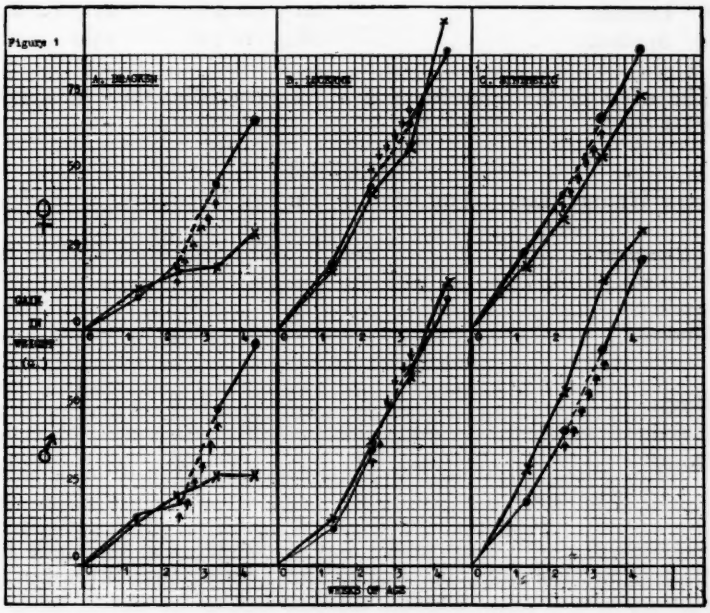


FIG. 1.—A comparison of the growth of rats receiving a basal ration plus "local" bracken, lucerne meal or casein and glucose. Each graph represents the mean of three animals. The arrows indicate daily injections of vitamin B_1

TABLE I

ANALYSIS OF LUCERNE MEAL AND DRIED BRACKEN USED IN THESE EXPERIMENTS

	Lucerne meal	Dried bracken	
		Local	Galashiels*
Dry matter	87.8	91.6	89.5
Crude protein	15.3	16.4	19.6
Ether extract	3.6	3.7	4.7
Crude fibre	24.6	25.9	14.3
Ash	8.8	6.0	5.8
Nitrogen-free extract (by difference) ...	35.5	39.6	45.1

In Table II the results are summarised to show the influence of B_1 on the growth rate of all rats.

TABLE II

WEIGHT INCREASES (G.) OF RATS FROM THE 17TH TO THE 24TH DAY OF THE EXPERIMENT; THE PERIOD IN WHICH 6 ANIMALS IN EACH GROUP RECEIVED B_1

* The greater proportion of frond to stem in the hand-picked Galashiels bracken is shown by the figures for crude protein, crude fibre and nitrogen-free extract.

Diet		Average gain in 7 days (standard error) No B_1 given	B_1 given
A	Bracken	7.9 (± 2.3)	28.3 (± 2.3)
B	Lucerne	20.5 (± 2.6)	21.7 (± 2.3)
C	Synthetic	28.2 (± 2.6)	25.0 (± 2.3)

The effect of the administration of vitamins other than B_1 is given in Table III.

TABLE III

WEEKLY WEIGHT GAIN (G.) OF RATS ON DIET A BEFORE AND AFTER DOSAGE WITH 5 B-VITAMINS OTHER THAN VITAMIN B_1

Treatment	Sex	Week before dosing	Week during dosing
Undosed	♂	1.2	2.4
Undosed	♂	6.2	—0.6
Undosed	♀	6.4	3.4
Average		4.6	1.7
Dosed	♂	—1.6	—1.0
Dosed	♀	6.8	8.2
Dosed	♀	4.6	—1.0
Average		3.3	2.7

Experiment 2.—The experiment was to compare the Galashiels bracken with the local bracken and to see whether after a longer term of bracken feeding any evidence of any other injurious material besides an anti-B_1 factor would appear.

Design of the Experiment.—Four litters of rats were used and 3 males and 3 females from each were taken for these experiments.

The rats were arranged in the following manner. Rats from each litter were numbered in order of weight as follows: males, 1, 2, 3; females, 4, 5, 6.

	Males			Females		
Litter 1	D*	E*	F*	E	D	E
„ 2	E	F	D	D*	F*	E*
„ 3	F	E	D	E*	D*	F*
„ 4	D*	F*	E*	F	E	D
Rat No.	1	2	3	4	5	6

Diet given D = Lucerne diet
E = Local bracken diet
F = Galashiels bracken diet

* Rats that received vitamin B_1 in addition to diet.

The diets were the same as in the first experiment except that sucrose was used instead of glucose and 40 parts of Galashiels bracken was substituted for the 40 parts of casein and glucose. Vitamin B_1 was given by subcutaneous injection on Mondays, Wednesdays and Fridays, 40 μg. being given on Monday and Wednesday, and 60 μg. being given on Friday.

Table IV gives the increase in weights for all rats after 21 days on these different treatments.

TABLE IV

GAINS IN WEIGHT (G.) FOR INDIVIDUAL RATS DURING THE FIRST 21 DAYS

Dosing with B_1	Diet D (lucerne)		Diet E (local bracken)		Diet F (Galashiels bracken)	
	♂	♀	♂	♀	♂	♀
No B_1	108	86	64	69	41	15
No B_1	73	79	39	55	10	35
Mean of 4	**86**		**57**		**28**	
Plus B_1 ...	98	77	105	86	80	74
Plus B_1 ...	104	95	92	53	82	50
Mean of 4	**93**		**84**		**72**	
"Dosed mean" minus "undosed mean"	**+7**		**+27**		**+44**	

S.E. of differences between means of 4 rats is 5.6 with 14 degrees of freedom.

The rats were allowed to continue on the same diets but deaths occurred in the group receiving diet F (Galashiels bracken) and no B_1 at the 35th and 36th days. The remainder of the rats on this diet were killed on the 43rd and 44th days as no more of this bracken was left. Rats consuming diets D and E were maintained on these rations for 96 days in all. The mean growth rates for 8 rats on each diet, four with and four without B_1, are given in Fig. 2. It is clear from the chart that the addition of B_1 to the lucerne diet made little difference to the well-being of the rats, for the slight increase shown in Table IV is not statistically significant.

The depression in the curve at the 64th day for those animals receiving B_1 is due principally to one animal who became sick at this period and lost weight from the 42nd to the 50th days. The rat subsequently recovered and her weight gains were normal although her weight lagged behind the others of this group.

The influence of B_1 in the group receiving diet E (local bracken) is most marked between the 16th and 32nd day. Here a marked depression in the growth curve is visible; later, however, the growth curve increases at the same rate as that for the rats receiving B_1, while in the last period it is more exaggerated so that there is no difference between them at 96 days. Galashiels bracken was clearly more potent than the local bracken; of the surviving two rats both were losing weight, and one which weighed only 11 g. more than at the start of the experiment would probably have died.

The growth curves for rats receiving local bracken (diet E) is reminiscent of curves obtained from refecting rats. As, however, the animals were caged in such a way that they could not consume their fæces, any adjustment as to the amount of B_1 obtained is likely to have occurred in the large gut itself.

Electrocardiograph records of the heart beats of each rat were taken on one

or more occasions. The individual variations in each group were considerable so that the results are not clear cut. The heart rates of the male rats receiving "local" bracken, however, on the four occasions in which they were recorded were slightly less than those found for the males of the same group receiving "local" bracken plus vitamin B_1.

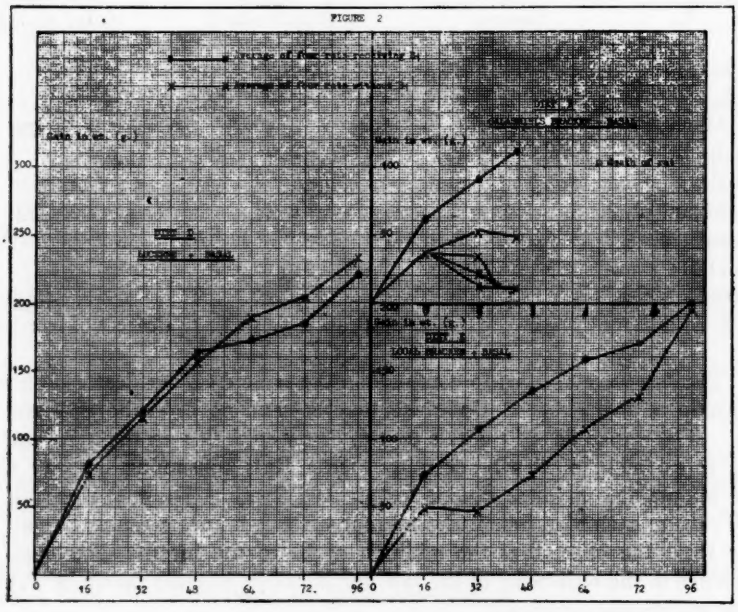


FIG. 2.—The growth rates of rats fed on a basal diet plus lucerne meal, "local" bracken or "Galashiels" bracken with and without the injection of vitamin B_1.

Post-mortem results (Dr. J. M. Naftalin).—All rats in the second experiment were examined *post mortem* and no macroscopic abnormalities were detected. The liver, kidneys, heart and sex organs from the rats of Group F (Galashiels bracken), with the exception of one rat receiving B_1, were sectioned and examined histologically. No abnormalities were detected in organs of the rats receiving B_1 except for a mild degree of degeneration in the tubular cells of the testes in one animal. In the three animals receiving no B_1 the liver cells were atrophied with small nuclei. The remaining animal, which was the best of the group, had a normal liver. In one animal that died the kidney cells were small and atrophied, and in the other rat that died there was marked degeneration of the tubular cells of the testes with occasional giant cells in the lumen of the tubule. These findings could be due to inanition alone.

2. EXPERIMENTS WITH PONIES

Two old ponies, Rose and Meg, after an initial period of hay feeding, were given local bracken with the hay. The quantity of hay was slowly reduced and

the quantity of bracken increased. Rose ate the bracken more readily than Meg whose consumption of bracken was irregular. The quantity consumed by each animal was measured as far as possible by weighing the food residues and their approximate consumption per day is given in Table V.

TABLE V

THE APPROXIMATE QUANTITIES OF HAY AND BRACKEN CONSUMED DAILY.

WEIGHT IN G.

Rose

Dates	Hay g.	Bracken g.	Body wt. lb.
Aug. 1-Sept. 10	2,722–4,086	—	480–444
Sept. 11-27	3,035	1,350	444–455
Sept. 27-Oct. 6	2,724	1,800	455–448
Oct. 7-12	2,250	2,250	448–441
Oct. 13-17	2,250	2,700	441–439
Oct. 18-Nov. 4	1,800	3,000*	439–445
Nov. 5-Dec. 5	1,350	3,150*	445–404

Meg

Dates	Hay g.	Bracken g.	Body wt. lb.
Aug. 1-22	2,724	—	634–567
Aug. 23-29	2,724	1,350	567–587
Aug. 29-Sept. 29	4,086	2,000	587–586
Sept. 30-Oct. 12	Food intake very variable		586–534
Oct. 12-17	2,724	600–2,520	534–517
Oct. 18-25	1,350	1,350	517–541
Oct. 26-Nov. 4	1,800	2,250	541–556
Nov. 5-Dec. 15	1,350	1,000–2,000	556–461

* Daily intake very variable.

Both ponies showed signs of bracken poisoning on three occasions; these consisted of a peculiar swaying movement of the hind quarters when walking and particularly when trotting, with marked disinclination to move and in particular to turn corners. On occasions co-ordination of movement was so disturbed that the ponies fell. When resting, the hind legs were straddled. Rectal temperature remained within normal limits; respirations were not increased but cardiac irregularities with an overall bradycardia were a common feature. The condition responded to B_1 given by injection or mixed with oats. In the latter instance the recovery was slower than after injection. Administration of B_1 in gelatin capsules by mouth had less effect and no real recovery occurred until a further dose was injected.

A summary of the development of the condition is as follows:—

Rose

Sept. 12.—Bracken feeding started. Thirty days later first symptoms observed.
Oct. 10.—Unwillingness to move, swaying gait, heart irregular.

Oct. 11.—Condition more pronounced.
Oct. 12.—Condition still pronounced.
Oct. 13.—Appetite still good but same signs observed.
11 a.m. given 40 mg. B_1 by subcutaneous injection.
11 p.m. condition improved; moves more freely.
Oct. 14.—Heart regular. Much brighter, willing to walk and turn round. Trotted willingly.
Oct. 15.—Normal.
After an interval of 19 days signs again appeared on November 3.
Nov. 3.—Signs seen again and become progressively worse. Only slight cardiac irregularities.
Nov. 5.—Film made of incoordinated walking.
2.50 p.m. given 10 mg. B_1 subcutaneously.
10.30 p.m. brighter and more active.
Nov. 6.—Pony greatly improved. Still some swaying seen.
Nov. 7.—Practically normal.
After a further interval of 14 days signs again appeared.
Nov. 21.—First signs seen. An irregular heart beat had been noticed for 5 days previously. The syndrome intensified and B_1 therapy was commenced on November 25.
Nov. 25.—100 mg. B_1 given in a gelatin capsule by mouth.
Nov. 26.—Some improvement but incoordination still marked. Irregular heart beat continues. Slight improvement continued until November 29.
Nov. 29.—Nearly normal but cardiac irregularities continue.
Nov. 30.—Swaying was still detectable. Cardiac irregularities continue.
Dec. 2.—Incoordination becoming worse again. Cardiac irregularities continue. The condition continued to deteriorate although cardiac irregularities disappeared on December 6 only to reappear on the 9th for 2 days. After December 11 no irregularities recorded.
Dec. 12.—No improvement. 20 mg. B_1 given subcutaneously.
Dec. 13.—Much improved.
Dec. 14.—Practically normal.

The condition responded quickly to the injection of vitamin B_1 even when only 10 mg. were given. The oral administration of B_1 in gelatin capsule so that no absorption could occur until the capsule disintegrated in the alimentary tract was far less effective even though 100 mg. were given. Recovery was not complete and the signs soon intensified again.

Meg

Aug. 22.—Bracken feeding started and the first signs were seen after 51 days.
Oct. 13.—The pony had seemed listless from October 9 but no cardiac irregularities were detected. On October 13, however, she developed the typical swaying gait, shown by Rose. The same syndrome developed during the next few days, and this pony showed the straddled stance of the hind legs more markedly than Rose. In

Oct. 13—*contd.* later stages muscular tremors were seen. Her heart, however, remained regular although the frequency of the beat was less than normal.

Oct. 18.—The condition is marked.

11 a.m. given 20 mg. B_1 subcutaneously. By 10 p.m. on the same day her condition was better, moved more easily in pen and seemed far less nervous.

Oct. 19.—Walks normally.

Oct. 20.—Appears quite normal; appetite very much improved.

After 15 days signs again appeared.

Nov. 4.—Cardiac irregularities noticed on October 30 and 31 and again on November 1. Clear signs of incoordination not seen until November 4.

Nov. 5.—Condition still the same. Film made of movements.

Nov. 7.—Worse; very unwilling to move. Fell in passageway. Twelve noon given 100 mg. B_1 in water mixed with $\frac{1}{2}$ lb. crushed oats. All eaten greedily.

10.30 p.m. still staggering when moved around her pen.

Nov. 8.—Rather more confident but still sways. No real improvement.

Nov. 9.—Cardiac irregularities reappear and continue until November 12.

4 p.m. no swaying when trotting. Considered to be normal as regards movement.

After 24 days a further attack occurred.

Dec. 4.—Heart normal and regular until on December 2 some irregularity was detected. This disappeared on December 3. On the 4th signs of swaying appeared.

Dec. 5.—Much worse. Given 100 mg. B_1 in a gelatin capsule by mouth.

Dec. 6.—No improvement.

Dec. 7.—Slight improvement but still very stiff and awkward.

Dec. 8 and 9.—Slight improvement maintained. Cardiac irregularities found on December 9.

Dec. 10.—Worse again.

Dec. 11.—No improvement. Cardiac irregularities detected.

Dec. 12.—Much worse. Given 20 mg. B_1 subcutaneously.

Dec. 13.—Much improved, still some swaying and heart still irregular.

Dec. 14 and 16.—Steady improvement. Bracken feeding stopped. Hay alone given.

Following the resumption of hay feeding the pony became acutely ill. She was very restless, sweating with increased respiratory and heart rates and a slight increase in rectal temperature. Her abdomen became tucked up in a way that was typical of grass sickness but she was never constipated. With careful nursing she recovered. Administration of a massive dose of B_1 subcutaneously had apparently no response. By December 25 she was clearly recovering and later she was normal again.

The heart rate.—Cardiac irregularities occurred in both ponies; they were

more marked in Rose than in Meg. An attempt was made on two occasions to record these with a Cambridge electrocardiograph, but the frequency of the beat is not sufficient to get enough records on to the length of paper that can be used to show the irregularity.

The intervals between beats with Rose were recorded approximately by timing with a stop-watch. The following figures illustrate the frequency of the irregularities.

Nov. 25.—Rose. Intervals between beats as timed on a stop-watch (in seconds):—

(a) 3 2 2 3 4 2 2 3 2 2 4 2 3 1 2 3 3 2
(b) 3 3 2 2 3 2 3 2 3 3 2 3 2 3 2 4 3
(c) 2 3 2 2 4 2 2 3 2 2 2 3
(d) 2 3 3 2 2
(e) 3 2 2 3 2 2

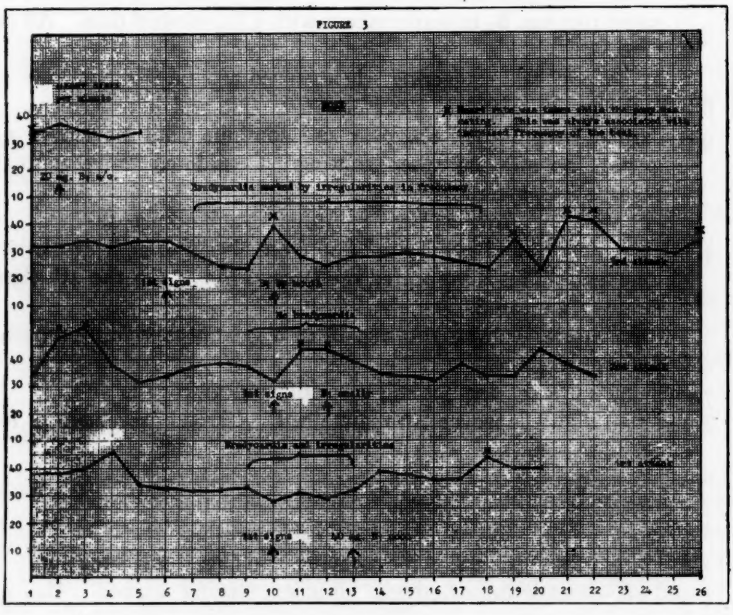


FIG. 3.—The variation in the frequency of the heart beat in the pony "Rose" in relation to the onset of visible clinical signs of "bracken staggers."

The impression obtained was that one beat in three or four was missing. This may be heart block rather than a missed beat. With Meg, whose heart rate was always slower than that of Rose, irregularities were not so common and when they occurred it appeared as though one beat in six or seven was missing.

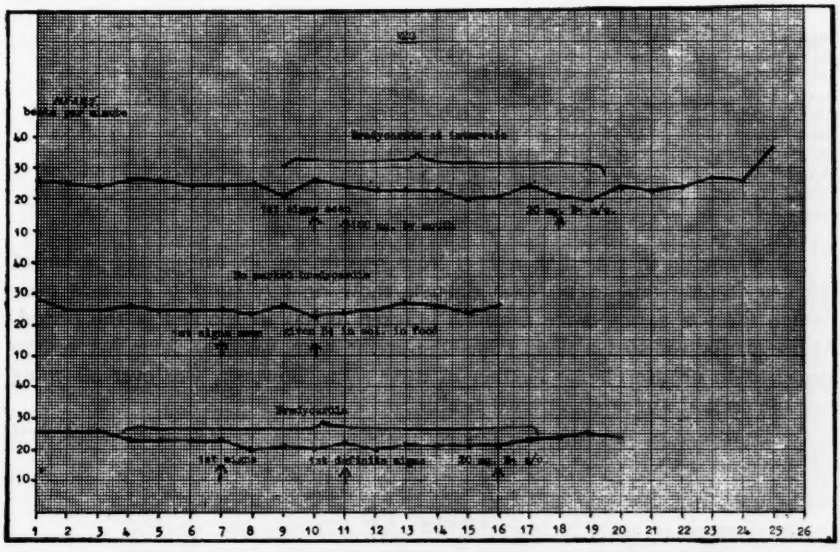
The occurrence of bradycardia and its relation to the visible signs are illustrated in Figs. 3 and 4.

Conclusions.—These experiments can be summarised as follows:—

1. Bracken staggers in ponies responded quickly to injection of B_1.

2. The response to B_1 in capsules by mouth was slow and not complete. Improvement was transient and the signs soon became more marked again.

3. Administration of B_1 in the food in one instance alleviated the conditions, although slowly. The possibility of absorption of B_1 in the mouth or œsophagus should not be overlooked.



Days

FIG. 4.—The variation in the frequency of the heart beat in the pony "Meg" in relation to the onset of visible clinical signs of "bracken staggers."

4. Bradycardia and cardiac irregularities accompanied the visible signs in the majority of instances, and this is compatible with B_1 deficiency. Cardiac irregularity may be the first sign to be detected.

As the animals survived no post-mortem was done. A sample of blood drawn from the jugular vein of Rose during one attack was examined by Dr. Howie and found to be negative for Pasteurella.

3. EXPERIMENTS WITH CATTLE

Two yearling heifers were fed hay and bracken in a similar manner to the ponies. Heart rate, rectal temperature and respiratory rates were recorded daily, together with food consumption. One of these animals consumed a considerable quantity of bracken over a long period and apparently thrived. She was later sold in the open market. The other animal developed what is held to be acute bracken poisoning and died.

The animal which survived did not eat the bracken readily and it was with difficulty that she was persuaded to increase the quantity consumed. In the

end she was eating twice the quantity daily that was eaten by the animal that died. The details of her food consumption and weight are given in Table VI.

TABLE VI

Black Heifer. Food Consumption in g.

Date	Hay (lb.)	Bracken (g.)	Date	Body weight (lb.)	Approx. proportion hay/bracken
July 8-11	10–11	80–425	July 11	553	23.0 : 1
July 11-26	6	350–2,525	July 24	528	1.3 : 1
July 27-Aug. 10 ...	9	1,460–2,655	Aug. 10	572	1.4 : 1
Aug. 11-19	10–11	1,975–3,430	Aug. 19	600	1.5 : 1
Aug. 20-Sept. 27 ...	12	1,600–4,050	Sept. 25	646	1.6 : 1
Sept. 28-Oct. 5 ...	10	3,410–3,820	Oct. 3	638	1.3 : 1
Oct. 6-16	9	4,275–4,950	Oct. 16	655	0.9 : 1
Oct. 17-26	8	4,730–5,400	Oct. 26	679	0.7 : 1
Oct. 27-Nov. 20 ...	7	5,520–7,080	Nov. 16	713	0.5 : 1
Nov. 21-Dec. 26 ...	6	4,450–7,440	Dec. 25	743	0.4 : 1

The animal increased in weight from 553 to 743 lb. during the period of bracken feeding and at no time did she show definite signs of sickness even though she was eating twice as much bracken as hay at the end of the period. At one period she had a slight discharge at the nose, while at times her rumen appeared to be overfilled. Neither of these signs, however, was ever alarming and the animal appeared in good health.

The details of the approximate food consumption for the animal that died are given in Table VII.

TABLE VII

"Blue" Heifer. Food Consumption and Body Weight

Date	Hay (lb.)	Bracken (g.)	Date	Body weight (lb.)	Approx. proportion hay/bracken
July 11-24	14–15	—	July 24	572	
July 25-Aug. 10 ...	9	2,700	Aug. 7	575	1.5 : 1
Aug. 11-19	9–12	2,210–3,600	Aug. 18	627	1.5 : 1
Aug. 28-Sept. 18 ...	12	2,675–4,500	Sept. 16	660	1.5 : 1

This animal remained in apparent good health until September 19, when the first signs of bracken poisoning were seen. At 3.30 p.m. she was found to have an increased rectal temperature.

From August 26 to September 18 rectal temperature had varied between 101.2 and 102.2° F. At 3.30 p.m. on September 19 it was 107° F. Heart rate, which had varied from 60 to 70, August 26 to September 18, was now 76. The respiratory rate was slightly, but not markedly, increased. The details of the syndrome from this time onwards were as follows:—

Sept. 20.—10 a.m. The animal lay down most of the time; a thick mucous discharge was running from the nose. No inclination to eat was observed, although she attempted to ruminate. No visible signs of rumen movement were seen, and there was a suggestion of abdominal

Sept. 20.—pain. Fæces were passed in small quantities and were looser than they had been previously.

2.45 p.m. Rectal temperature 107.5° F.

5.45 p.m. No improvement. 45 mg. B_1 given intramuscularly.

Sept. 21.—7.30 a.m. Rectal temperature 106.4° F. Respiratory rate 30/m. Heart rate 96/m.

11 a.m. 45 mg. B_1 given intramuscularly. Animal drinking water.

11.45 a.m. Seen to be ruminating.

Sept. 22.—10.30 a.m. Rectal temperature 107.2° F. Respiratory rate 32/m. Heart rate 30/m.

The urine was found to be full of blood. After spinning in a centrifuge a thick deposit of corpuscles was obtained; no hæmoglobin in solution was present, for the fluid was a clear yellow colour giving a positive Benedict reaction and a positive reaction for albumin. The cellular deposit proved to be largely intact red cells when seen under a microscope, and there were no signs of pus or pathogenic bacteria. A few nucleated pear- or round-shaped cells were seen, which were probably epithelial cells of the bladder. No casts were observed.

3.15 p.m. The animal was examined by Dr. S. Jamieson, Veterinary Investigation Officer for the North of Scotland College of Agriculture. Petechial hæmorrhages were seen in walls of vagina and a small fleck of blood in the nasal discharge. For the first time blood in the form of large black clots was seen in the fæces, suggesting hæmorrhage high up in the intestine. Dr. Jamieson's opinion was that the signs were typical of those of acute bracken poisoning. Later in the afternoon 100 mg. B_1 given intravenously.

Sept. 23.—The animal was alive at 9 a.m. but dead at 11 a.m. The carcase was taken to Dr. Jamieson's laboratory for post-mortem examination. Extensive hæmorrhages on the peritoneal surface of most of the abdominal organs were found. A hæmorrhagic cystitis was present and much hæmorrhage had occurred into the intestine. Dr. Jamieson provided the following report:—

Subcutaneous tissues.—Considerable ecchymoses and larger areas of hæmorrhage.

Thorax.—Contained approximately 1 pint of reddish-coloured pleural fluid.

Trachea.—Inflamed throughout its length with considerable hæmorrhages under the serous coat.

Lungs.—A few small areas of pneumonia.

Heart.—Ecchymoses on the myocardium, hæmorrhage on the inner wall of left ventricle.

Peritoneum.—Peritoneal cavity practically full of blood.

Reticulum.—Extensive hæmorrhage over whole surface under serous coat.

Rumen, omasum and abomasum.—Patches of hæmorrhage under serous coat.

Small and large intestine.—Extensive hæmorrhage under the serous coat. The former showed ecchymoses on the mucous coat, with some ulceration and hæmorrhage into lumen. There was also a considerable hæmorrhage into the lumen of the large intestine and cæcum.

Liver.—Normal, except one patch of hæmorrhage under serous coat.

Gall bladder.—Greatly enlarged, with considerable sub-serious hæmorrhage.

Kidneys.—Normal, except for very slight patches of inflammation.

Bladder.—Extensive sub-mucous hæmorrhage, and full of hæmorrhagic urine.

Lymph glands.—Enlarged and congested throughout the body.

Bacteriological examination of the spleen, small intestinal contents and bladder wall of the heifer revealed no bacteria of any significance. Intravenous inoculations of bowel filtrates into mice caused no deaths. Examinations of sections made from the small intestine, liver, kidney and bladder wall revealed in all considerable hæmorrhage.

Discussion

The experiments with rats and ponies support previous work of Weswig *et al.* (1946), Evans & Evans (1949) and Thomas & Walker (1949) which indicate the presence of an anti-thiamine factor in the bracken; the condition produced in both rats and ponies responded to the administration of vitamin B_1 and there is no reason to doubt that "bracken" poisoning in these animals is in fact an induced thiamine deficiency.

The position with cattle is different for the following reasons: the onset occurred after a considerable period of feeding bracken during which time no signs of debility or any other symptom of vitamin deficiency were noticed; the high temperature that developed suddenly and the acute course of the illness bears little resemblance to the picture seen in rats and ponies. Finally, injection of vitamin B_1 did not cure the condition.

It is important to mention that in "Chastek" paralysis in foxes, which is an induced thiamine deficiency due to feeding raw carp which is rich in an anti-thiamine factor, hæmorrhages into the myocardium and sometimes into the small intestine may be seen on post-mortem examination. Feeding vitamin B_1 prevents the condition, but the evidence of the curative value of B_1 in acute cases is not well established (Green, Evans, Carlson & Swale, 1942). There is, therefore, some similarity between this picture and that of bracken poisoning in cattle.

The interesting feature of the cattle experiment is that only one of the two animals developed bracken poisoning. On September 18 when the heifer that died was last in health it had eaten 417 lb. of bracken in 55 days together with 608 lb. of hay, an average of $7\frac{1}{2}$ lb. a day, while the surviving heifer had eaten 458 lb. of bracken in 76 days together with 904 lb. of hay, an average of 6 lb. a day. Even so, the surviving animal later on consumed daily considerably more than $7\frac{1}{2}$ lb. of bracken daily but remained unaffected. One can only suspect

that the slow rate at which the quantity of bracken consumed increased, allowed the animal to adjust itself to the bracken and it is possible that some of this adjustment occurred in the rumen.

This hypothesis is somewhat strengthened by the fact that in the second experiment with rats which were given local bracken the initial depression of the growth rate was overcome later in a manner reminiscent of refecting rats. This is a clear indication that some means of combating the induced vitamin B_1 deficiency had developed. There is no clear conception of the nature of this process for even if we assume that bacteria synthesising vitamin B_1 in the rumen or large gut become resistant to the anti-B factor, it is hard to visualise how synthesised vitamin B_1 escapes it. Thomas and Walker (1949) found that the anti-factor was still present in the fæces of rats fed bracken and presumably B_1 must escape from the bacterial cell somewhere in the alimentary tract before it can be absorbed by the animal. It may be that bacteria capable of destroying the anti-factor develop under certain conditions both in the rumen and in the large intestine.

The greater activity of the " Galashiels " bracken, gathered from a hill where bracken poisoning had been reported previously, in comparison to local Aberdeenshire bracken may not be a true effect for it was hand-picked and contained more frond and less stem than local bracken, as is shown by the analyses given in Table I, and was not dried in quite the same fashion or as rapidly. It is clear that the anti-factor was present in bracken grown in both areas in sufficient quantity to produce a B_1 deficiency in rats and ponies. For experimental purposes, therefore, it is unnecessary to gather bracken from areas where clinical cases of bracken poisoning have been reported.

Summary

1. Bracken cut in June, after chopping in a silage cutter and drying in a grass drier, contained a factor that induces B_1 deficiency in rats and ponies. Both Aberdeenshire and Galashiels bracken contained this factor although bracken poisoning has not been recorded in the Aberdeenshire area.

2. Rats fed 40 per cent dried bracken did not gain weight normally and some ultimately died. Treatment with vitamin B_1 allowed normal growth rates to occur. The growth rate in one group of rats fed Aberdeenshire bracken after an initial depression later returned to normal without administration of B_1. Other B vitamins do not correct the deficiency.

3. Two ponies fed dried bracken and hay developed on three occasions a condition that was characterised by incoordination of movement and bradycardia due to irregularities of the heart beat. Treatment with vitamin B_1 by subcutaneous injection cured the condition. Administration of B_1 in a gelatin capsule by mouth was less effective.

4. One of two heifers eating dried bracken and hay developed a condition similar to bracken poisoning. Injection of vitamin B_1 did not cure the animal and it died. The post-mortem picture was consistent with that described for

bracken poisoning and no pathogenic organisms which could account for death were isolated from the carcase. The other heifer remained in apparently good health although ultimately consuming more bracken daily than the one that died.

Acknowledgments

We wish to thank Dr. S. Jamieson and his staff for the pathological and bacteriological examinations made on the heifer that died, Dr. J. M. Naftalin for post-mortem examinations of the rats, Mr. J. Davidson for analyses of the lucerne and bracken meals, and Mr. R. S. Reid and Mr. G. Porter for their records of the rats used in these experiments.

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REVIEW

DISEASES OF THE PIG, by D. J. Anthony. Third edition. Pp. xii + 309 with 53 Figs. Price 17s. 6d. London: Baillière, Tindall and Cox.

ANTHONY'S book on the diseases of the pig was first published in 1940. This was a most fortunate circumstance for at that time, owing to the exigencies of war, many people, who were not experienced in the care of this animal, were taking up pigkeeping. The book was a "godsend" not only to these new pigkeepers but also to veterinary surgeons whose advice was sought on problems connected with housing, feeding, diseases, and even on the choice of animal. The book soon became a standard reference and a second edition was required in 1949. Since the war and especially in recent times, owing to some easement of the food supply, the commercial or large pigkeeper is rapidly increasing his stock and unless the situation be carefully watched there may be a return to the "wholesale" disease problems which beset us before the war and which, owing to the war-time emphasis on the smaller, more isolated "backyard" unit, have shown such a welcome decline during the last ten years.

How can this be averted and at the same time how can we increase the pig population so that as a source of food they will fulfil our requirements? The answer is in better breeding, housing, feeding and hygiene and a better appreciation of any deviation from normal health.

Anthony sets out to meet these requirements and in the limited compass of approximately 300 pages we think that he has gone a long way to success.

The title of the book does not indicate its scope for there are most valuable chapters on breeds of pigs (with illustrations), requirements for the markets, pork and bacon, housing and management, feeding, dentition and restraint. The chapter on housing and management is done well and British pigkeepers will find the plans and illustrations of the Scandinavian type of "pig house" extremely useful.

In the chapter on feeding, the section on emergency foods will be appreciated for until world supplies of cereals become normal again there will be many occasions when the pigkeeper can usefully employ alternative foods. The author also gives a short account of the Lehmann feeding system which in short amounts to this: "A basic ration of high-quality food supplemented by some bulky foods such as potatoes, swedes, silage, etc., the basic food maintaining good health and the bulky foods providing for the fattening."

The part of the book which deals with disease first considers scheduled disease. Then we have chapters on constitutional and local disease. Deficiency diseases and diseases caused by animal parasites each have a chapter.

Poisons and poisonous plants are also reviewed. This edition has a new chapter, namely, that on post-mortem methods. Such information can be extremely useful, but we do not think that this chapter is up to the author's standard. It lacks detail; for instance, the examination of the heart is encompassed in 20 words. The gastrointestinal tract is evidently just cut open and

examined, and running water is an advantage. It may be a disadvantage and wash away things one should see. It is also advisable to instruct the would-be "post-mortem" man to open the gut along the mesenteric border if he wants to get a good view.

In spite of these few criticisms this is a good book and the veterinary surgeon, the student and the pigkeeper will profit greatly from its perusal. Those who would delve further will find the list of references on pages 295/6 valuable.

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